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The oral care for children with malignancies

Winning 2003 Postgraduate Essay
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'if he (sic) is able to tell his patients about their past and present symptoms…also…what is going to happen, as well as …the details they have omitted, … people will have no qualms in putting themselves under his care'. ***(Hippocrates of Cos, c.450BC)***

Introduction

Childhood malignancies are rare, yet represent the most common cause of death in children under 15 years old.[1] Treatment for these conditions have improved markedly with newer methods of diagnosis, staging and therapy resulting in more long-term survivors. For example, in acute lymphoblastic leukaemia, a five year disease-free survival rate of over 90% has been reported.[2]

Malignancies most commonly affecting children, such as leukaemia, brain tumour, lymphoma, neuroblastoma, rhabdomyosarcoma and Wilms' tumour, differ from those seen in adults.[1,3] Complications from malignancies frequently occur in the oral cavity, and intensive therapeutically-successful treatment may be associated with increased morbidity.[4,5]

With regard to oral care for children with malignancies, two main aspects will be discussed in this essay. Firstly, oral care during treatment of the malignancy per se, and secondly, oral care required during different treatment modalities, complications and immunosuppression.[3] Severe complications may require cessation of therapy.[6] Appropriate management of such complications is essential and should be carried out by an oncology team including a dentist.[5,7]

Treatment of malignancies in children

Treatment of childhood malignancies is complex, typically undertaken in a multidisciplinary facility. Direct treatment of malignancies involves chemotherapy, radiotherapy and in the case of solid tumours, surgery.[3] Bone marrow transplantation (BMT) is used in the treatment of some acute and chronic leukaemias, recurrent lymphomas and some solid childhood tumours such as advanced stage neuroblastoma.[1,8]

Chemotherapeutic agents have selective toxicity towards rapidly dividing cells based on their interference of the cellular cycle.[9] Chemotherapy should only destroy the malignant cells without affecting normal cells. However, most drugs are not sufficiently specific and damage other high turn-over tissues, such as skin, hair, mucous membrane and the haemopoietic system.[3,10]

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Radiotherapy plays an important role in the treatment of lymphomas, brain tumours and many solid tumours. Cranial irradiation can eradicate malignant white cells in the central nervous system which are protected from chemotherapy by the blood-brain barrier.[10,11] Radiotherapy aims to

THIS ISSUE

President's Report

As this is to be my last presidential message I have decided to briefly express some personal observations and thoughts regarding our Society. I have always enjoyed ANZSPD activities over the last 20 years. I have found members that I have met, both initially within my state branch, and later also at Biennial Conferences to be mostly congenial and friendly. I now count many as friends.

Personal qualities of warmth and friendliness amongst members reflect empathetic qualities needed for successful dental care delivery for children. Such qualities are needed in equal measure with up to date scientific knowledge and technical skills. Our Society is I believe the main organisation that provides high quality continuing education in paediatric dentistry; a process that is painless and dare I say enjoyable, as well as promoting harmonious interaction between those involved in service delivery, teaching and research into promoting child dental health throughout Australia and New Zealand.

By the time this is in print our 14th Biennial Conference will be upon us. I look forward to meeting with our widely scattered members here in Melbourne, as well as welcoming newcomers to our fold. Our local organising committee consisting of Mala Desai, Jodie Heap, Karen Kan, John Sheahan, Donna Tomeski, Felicity Wardlaw and myself, with input from Nicky Kilpatrick and Louise Brearley, have strived to make this 14th Meeting an unforgettably rewarding event. We hope that you find the 14th ANZSPD Conference to be both educationally informative as well as providing enjoyable collegiality.

I take this opportunity to thank Alistair Devlin, our perennially hard working Secretary/Business Manager for keeping the Society and this President on track, and also Karen Kan as Editor, without whose largely unseen efforts recent issues of "Synopses" would not have been published.

Finally I wish to acknowledge the substantial and consistent long-term support given to both ANZSPD educational activities and to publication of "Synposes", by Colgate Oral Care and Dr Jackie Robinson; which I sincerely hope will continue in years to come.

Chris Olsen

Abstracts from the 9th Australasian Academy of Paediatric Dentistry Scientific Meeting held at Manly Pacific Hotel, NSW, on Saturday 13 September 2003

An interesting case

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ABSTRACT

The avulsion of permanent teeth has its highest incidence in the 7-9 year old age group, who have newly erupting incisors and resilient bone. Avulsion is particularly problematic in terms of medium and long-term treatment. The pulps of such teeth, in the majority of cases, ultimately progress towards pulpal necrosis. Although normal periodontal healing has been reported to occur in 20-26 percent of replanted permanent teeth in experimental studies, such figures require replantation under optimal conditions, and within five minutes of avulsion. Typical periodontal responses include surface resorption and replacement resorption. Periodontal healing and the avoidance of the resorptive process is related to the vitality of the periodontal ligament, which is in turn closely related to the length of time the tooth has been out of the socket. Various approaches have been proposed minimise such resorption. Endodontic treatment, particularly with the use of calcium hydroxide medicament, has been shown to be effective in the reduction of inflammatory root resorption. The application of fluoride prior to replantation has also been found to result in a reduction in the progression of root resorption. Immediate extraoral root canal treatment at the time of replantation is documented, but is technically demanding, complex and time consuming. Endodontic stabilisers (endodontic posts) provide a simpler method of achieving canal obturation outside of the mouth, prior to the replantation of an avulsed tooth.

A case report is presented, involving a 13-year-old boy with a complex cardiovascular history, asthma, and behavioural problems. The upper right central incisor was avulsed several hours earlier and storage was in the mouth. Hypodontia and the necessity for treatment under general anaesthesia in a single visit ruled out conventional treatment in this case. Replantation this avulsed central incisor is described with the use of an endodontic stabiliser.

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deliver lethal doses of radiation to the tumour whilst minimising the dosage to surrounding tissues. When cranial radiation is required, irradiation of the eyes and orodental structures is minimised.[3,12]

Head and neck tumours of the maxillofacial region are treated principally with surgery.[13] Resection of the maxilla for the treatment of an osteosarcoma, for example, produces deficits and cosmetic deformities. Consequently, when a maxillectomy is required for treating a childhood malignancy, a maxillofacial prosthodontist must be included in the pre-surgical assessment.[13,14]

BMT is used for treating diseases affecting the bone marrow directly or indirectly.[15] Preoperative conditioning involves high dose cyclophosphamide therapy and total body irradiation, aiming to destroy the patient's bone marrow and replace it with a transplant of healthy cells.[3] Donors can be identical twins, HLA identical or near-identical siblings, other family members, unrelated but compatible donors (allogenic), or the patient's own bone marrow (autologous) harvested during disease remission and stored for re-infusion.[8]

Oral complications of cancer treatment

Complications frequently develop in the oral cavity either as a result of malignancy or as a side effect of therapy.[4] The prevalence of oral complications varies, with some authors suggesting complications are more common in children affecting up to 90% of paediatric oncology patients.[5,16] The increased mitotic index in children may account for greater susceptibility to side effects of therapy.[16]

Complications associated with chemotherapy

Many chemotherapeutic agents implicate oral complications, producing stomatotoxicity either directly or indirectly. Direct stomatotoxicity follows cytotoxic action on oral mucosal cells, leading to inflammation, thinning and ulceration of the mucosa.[17] Mucositis occurs in up to two-thirds of paediatric oncology patients, especially during induction and intensification phases of chemotherapy when high doses of multiple chemotherapeutic agents are administered.[3] Oral mucositis may cause acute pain, dysphagia and malnutrition. The onset is usually within 3-7 days of chemotherapy initiation and the duration varies from several days (single agent therapy) to several weeks (combined agent therapy).[18] Mucositis resolution occurs 7–14 days after onset, paralleling haematological recovery. Chemotherapy has fewer long-term effects on the salivary glands, although these patients may experience temporary changes in salivary quantity and quality.[19]

Indirect stomatotoxicity results from the effect of chemotherapy drugs on cell populations other than the oral mucosa. Bone marrow suppression results in thrombocytopenia and neutropenia which in turn lead to abnormal bleeding and lowered resistance to infection.[3] In the thrombocytopenic patient, slight trauma caused by oral hygiene procedures or eating can cause bleeding apparent as mucosal petechiae or ecchymoses and bullae formation in more severe forms.[20] Persistent gingival oozing and marginal gingivitis is common. Recent extraction sockets may bleed severely.[3]

Most cytotoxic drugs are myelosuppressive, increasing the susceptibility of an already debilitated patient to infections with bacteria, fungi and viruses.[3] Cytotoxic effects reduce local immunity and increase microbial accumulations on the mucosa. Mucosal integrity may be impaired; together with increased plaque and gingival inflammation this facilitates systemic entry of commensal bacteria.[21]

Acute dental infections may arise, either as a result of periodontal disease or subsequent to pulp necrosis. Non-vital deciduous teeth with chronic periapical infections are prone to becoming acute, resulting in septicaemia. Generalised chronic marginal gingivitis, pericoronitis, oral and perioral pathosis due to Candida and herpes simplex viruses may occur. Opportunistic microbes readily infect oral lesions, and such secondary infections may have serious or even fatal consequences.[12] In severely compromised patients, over 50 percent of systemic infections result from oropharyngeal microorganisms.

Complications associated with radiotherapy

The effects of radiotherapy are localised, additive and cumulative, depending on the dosage, site and cell sensitivity.[22] When the mouth is in the radiation field, initial inflammation is followed by sloughing and ulceration which is more severe about 2–4 weeks after radiotherapy commences and resolves in a further 2–3 weeks.[3] In this period, the patient suffers pain and difficulty eating. When the radiotherapy field involves major salivary glands, hyposalivation and xerostomia frequently occur, and saliva becomes more viscous and acidic.[10,23] Hyposalivation may be permanent, with partial recovery after many months. Loss or alteration of taste follow xerostomia, and may also result from direct damage to taste buds.[3] To alleviate mouth soreness and dryness, patients will change to a softer, more cariogenic diet including sweet, acidic fruit drinks. Teeth become sensitive and difficult to clean. These changes together with a cariogenic flora promote radiation caries, which may become apparent 2–12 months after radiotherapy. Lesions appear at the cervical margins of teeth and can rapidly destroy the whole dentition.[3]

Xerostomia potentiates oral infections.[3] Suppressed oral immunity and compromised barrier mechanisms lead to mucosal ulcers providing an entry portal for fungi and bacteria. Compared with adults, the salivary gland tissue in prepubertal children seems more resistant to radiotherapy and chemotherapy. The severity of salivary and mucositic complications is less than experienced by adults and adolescents when exposed to comparable doses of fractionated total body irradiation.[19]

Complications associated with bone marrow transplantation (BMT)

BMT involves chemotherapy and sometimes total body irradiation, making patients prone to the above-mentioned complications.[15] Almost all children undergoing BMT develop oral mucosal changes, typically hyper-keratinization and erythema, occurring

maximally 4–14 days after transplantation. Mucosal atrophy is often seen 1–3 weeks after BMT, severe oral pain often requiring narcotic analgesia.[3,15] Salivary immunoglobulin production, part of the mucosal immune defence, is impaired following BMT perhaps due to the total body irradiation conditioning prior to transplant.[24] Subsequent opportunistic infections are major causes of morbidity following BMT and are the primary cause of death in patients with graft versus host disease (GVHD).[25]

GVHD occurs in allogenic transplants, where transplanted immunologically active T-lymphocyte 'graft' cells recognise immunocompromised 'host' tissues as foreign.[3] Both acute and chronic forms still cause considerable morbidity and mortality and constrain successful bone marrow transplantations.[26] The acute form, occurs early post-transplant, includes fever, diarrhoea, muscle wasting and abnormal liver functions leading to jaundice. The chronic form, which may follow the acute form, or occur several months later, is characterised by lichenoid- or scleroderma-like changes of the skin, keratoconjunctivitis, abnormal liver function and intestinal problems. The oral manifestations vary with the severity of the conditions: painful oral mucosal desquamation and ulceration occur in the acute form, while mild oral mucosal erythema, desquamative gingivitis, loss of lingual papillae, lichenoid lesions and xerostomia occur in the chronic form.[3]

Managing the oral complications of malignancy treatment

Combined chemotherapy and radiotherapy show three times more oral complications in children than adults.[5] The type of malignancy and location, dose and protraction of radiation, degree of oral care before and during treatment and the developmental status of the patient all influence the type and severity of oral complications.[5] The oral mucosal problems can be severe and prevent the child from eating and drinking and require pain relief.[19] Adequate oral care can do much to alleviate the problem.[11]

The management of oral mucositis and ulceration is largely symptomatic. Patients should be advised to eat soft, bland food and avoid irritants such as smoking, spicy and acidic foods such as salt and vinegar crisps. Routine management consists of topical mouthrinses containing various agents with palliation being the ultimate goal.[5] A randomised control trial showed the three common mouthrinses (chlorhexidine, salt/soda and lidocaine/Benadryl/Maalox known as 'magic') to be equally effective.[27] Benzydamine hydrochloride (Difflam) oral rinse or lozenges can relieve the pain of mild to moderate oral and pharyngeal mucositis. This drug is valuable in relieving discomfort before meals, but is not recommended for young children.[3] Xylocaine topical preparation (2%) is also useful, especially for patients with more severe mucositis and ulceration, but needs to be used with care as the mouth and throat can be numbed and the cheeks and tongue bitten accidentally.[3] The use of antihistamines, topical anaesthetics and antacids, alone or in combination, have been suggested without experimental evidence to support the efficacy of one over another.[15] For young children, promethazine hydrochloride (5mg/5ml elixir Phenergan) can be used as a topical anaesthetic for painful ulcers. The recommended dosage must not be exceeded; the sedative effect may be advantageous.

Herpes simplex virus (HSV) is the most common viral infection associated with chemotherapy and BMT. Herpes labialis (single or multiple cold sores) is the most common presentation; less frequently a generalised ulcerative stomatitis similar to the primary lesion occurs. Rarely, chronic destructive mucocutaneous herpes simplex with systemic involvement occurs. Acyclovir (topical, oral or intravenous forms) is the drug of choice.[3] Prophylactic acyclovir should be considered for patients seropositive for herpes virus and at high risk of reactivation.[5] As HSV infections sometimes present atypically, swabs of any suspicious lesions should be taken for virology. A rising antibody titre over ten days confirms an HSV infection.

Oral bacterial infections in chemotherapeutic patients occur secondary to mucosal ulceration following therapy-related neutropenia. The clinical appearance of inflammation can be reduced or absent in immunocompromised patients, complicating identification and management. Lesions should be cultured before prescribing antibiotics, when possible. Chlorhexidine rinse is effective in reducing mucositis in children receiving BMT and chemotherapy.[5]

Candidiasis is the most common fungal infection in children during severe immunosuppression and neutropenia. Extensive use of broad-spectrum antibiotics, chemotherapy-associated immunosuppression, inadequate oral hygiene and nutrition, and poor physical condition increase the risk of oral candidiasis. In children, candidiasis occurs mostly on the buccal mucosa, tongue, gingiva and pharynx. Cultures should be obtained for diagnosis and treatment of immunocompromised patients.[5] Nystatin suspension is commonly used to treat oral candidiasis; 1ml of sugar-free 100,000 units/ml Nystatin mixture is rinsed in the oral cavity for a minute and swallowed, at least four times daily as a prophylactic measure.[3] Fluconozole and Amphotericin B are reserved for chronic and serious life threatening infections.[19] Chlorhexidine and Nystatin use should be staggered as in combination they are ineffective against Candida.[5]

Bleeding from gingiva and oral mucosa are commonly associated with chemotherapy, reflecting the severity of thrombocytopenia, neutropenia and immunosuppression. Gingival bleeding is exacerbated when oral hygiene is poor. Although immaculate oral hygiene measures are recommended, patients with low platelet counts may not be able to brush, therefore rinsing and swabbing the mouth with chlorhexidine is preferred.[5] Spontaneous gingival bleeding occurs with platelet counts of $15x10^9/l$ or less and is especially bothersome in children with mobile primary teeth.[19] Management of oral bleeding may require topical tranexamic acid.[6] Platelet transfusion is undertaken when local measures are unsuccessful. Recombinant granulocyte-colony stimulating factor is sometimes used to improve neutrophil recovery following cancer treatment.[28]

Xerostomia commonly follows radiation to the head and neck region.[29] The saliva becomes thick and viscous and mastication, swallowing, speaking and taste become impaired. A diet rich in sucrose and carbohydrates associated with poor oral hygiene

predisposes radiation dental caries.[5] Optimal oral hygiene is vital, especially frequent rinsing.[15] Managing xerostomia involves combined strategies including synthetic salivary substitutes, stimulation of remaining salivary tissues by chewing gum and use of topical fluoride.[5] Recent studies with casein derivatives complexed with calcium phosphate have shown promise of caries prevention in patients with salivary gland dysfunction.[30]

Dental assessment and management prior to treatment of malignancy

Ideally, all children should be dentally fit prior to commencement of treatment for the malignancy.[8] However, due to the child's medical status, this is not always possible.[6] Screening for dental pathology should take place at the earliest opportunity in order to minimise the risk of infection and haemorrhagic conditions and to identify existing pathology which may be dormant, but can be exacerbated by immunosuppression.[3,19] Abscessed, grossly carious or exfoliating teeth should be removed at least ten days before the start of therapy, especially where head and neck radiotherapy or BMT is planned.[8] Other carious teeth should be temporarily restored and non-urgent operative treatment delayed until the child is in remission.[19] Vital tooth pulpotomies on primary molars are contraindicated as treatment failure carries a risk of serious infection.[8]

The objective is to minimise oral foci of infection and most hospitals have oral care included in their treatment protocols.[31] The importance of maintaining a high standard of oral hygiene in the child should be stressed to the parent. Toothbrushing with a soft brush should be continued throughout therapy although occasional relief due to severe gingival bleeding or stomatitis is understandable. Flossing is recommended unless the platelet count is low, when instead the teeth and mouth should be cleaned with soft sponge sticks. Toothbrushing must recommence at the earliest possibility.[3]

The routine use of an antimicrobial mouthrinse such as 0.12% chlorhexidine is a valuable preventive measure in reducing the oral bacterial load, preventing secondary infections, and suppressing Candida.[32] Chlorhexidine appears to modify the course of mucositis and reduce the risk of systemic infections of oral origin.[28] When rinsing is not possible as with a younger child, oral swabbing is recommended.[3] Antifungal lozenges or sugar-free suspensions (eg. Nystatin) are also part of many protocols. For children or adolescents with high caries risk, or living in a non-fluoridated area, toothpaste with a higher fluoride concentration may be recommended such as neutrofluor 5000™ or adult toothpaste (1,500ppmF). Topical fluoride gel is recommended for BMT and radiotherapy patients at greater risk of xerostomia to stimulate remineralisation and reduce bacteria.[11]

Oral care management during treatment for malignancy

When treatment starts, the transient xerostomia associated with chemotherapy and total body radiation reduces salivary flow. Saliva becomes ropy and viscous, causing difficult and painful swallowing, a shift in the normal oral microflora and an increase in dental caries.[33] Diet, appetite and taste changes occur; parents and nutritionists struggle to maintain the child's caloric intake and to counteract nausea and vomiting. Vomited stomach acids irritate the mucosa and decalcify the teeth, promoting dental erosion.[34,35] Remineralisation with casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) may be possible. A new oral care product, Recaldent™, is available as a water-based sugar-free crème (Tooth Mousse, GC Corporation, Japan) and a chewing gum. When applied to tooth surfaces, CPP-ACP binds to the plaque biofilm. Under acidic conditions, CPP-ACP releases calcium and phosphate ions enhancing remineralization and significantly reducing caries risk. CPP-ACP remineralises white spot lesions and acts synergistically with fluoride in remineralising erosion.[36,37]

Dental treatment required for a child with malignancy, must be discussed with the child's oncologist or haematologist. When possible, routine procedures should be carried out in a dental chair, but children requiring extensive treatment may need treatment under general anaesthesia.[8] A recent full blood count is essential, including a differential white blood and clotting profile. Ideally, an absolute neutrophil count exceeding $1x10^9$/L and a platelet count of at least $60x10^9$/L are considered acceptable for oral surgery, provided these counts are anticipated to remain stable for 14 days.[38]

If acute infection is present, cultures must be taken from venous blood and from any pus or exudates. Consultation with an infectious diseases specialist is recommended to choose the appropriate antibiotic before the results are available.[8] For acute dental infections, the most predictable dental treatment (often extraction) should be contemplated considering the medical risk-benefit ratio.[38] If extractions are required, the platelet count should exceed $80x10^9$/L and transfusion is required for levels below $50x10^9$/L.[3,6] The neutrophil count should be at least $1x10^9$/L to $1.5x10^9$/L, and then only with concurrent administration of prophylactic antibiotics.[28] A resorbable haemostatic agent, Surgicel™ is recommended and the sockets compressed and sutured with soluble sutures. An antifibrinolytic agent such as tranexamic acid can promote haemostasis. After the treatment, suitable antimicrobial therapy should be continued during healing until the neutrophil count has recovered.[3]

Management of the long term effects of malignancy treatment

Modern cancer treatments are improving the prognosis of malignancies and patients are surviving longer with the late effects of some treatments appearing.[39] These include caries, xerostomia, alteration to the developing craniofacial skeleton and effects of arrested dental development.[19,40] Chemotherapy interferes with cellular cycles and intracellular metabolism and causes retarded dental development, microdontia, agenesis, enamel hypoplasia and discoloration and root malformation.[9,41,42] Radiotherapy has direct effects on bone, soft tissue and blood vessels and profound effects on rapidly growing children resulting in bony hypoplasia, orofacial asymmetry and malocclusion.[10] Arrested tooth development will depend on the anatomical position, dose and intensity of the treatment, and the child's age.[42] Particular effects of radiotherapy include destruction of the tooth germ, failure of tooth development, stunted growth of crown

and root, incomplete calcification and hypoplasia of enamel, premature root apexification and root tapering.[10,19,22,39] One of the indirect effects of radiotherapy to the central nervous system can result in diminished production of growth hormone and thyroid stimulating hormone, adversely affecting craniofacial development and odontogenesis.[10,22]

A comparative study on the dento-facial development of children who were long-term survivors of acute lymphoblastic leukaemia (ALL) treatment revealed that those treated before the age of five years and who received chemotherapy plus radiotherapy had more severe dental abnormalities (tooth agenesis, arrested root development, microdontia and enamel dysplasia) than those treated with chemotherapy alone.[43,44]

Children with malocclusions due to disturbed dental development and altered facial growth following therapy may need orthodontic rehabilitation. Orthodontic treatment is often possible and without side effects, but lighter forces and compromised treatment plans may be used in view of the previous medical condition.[45] Orthognathic surgery such as bone grafting and osteotomies to improve facial deformities have a high risk of osteoradionecrosis.[22,43]

Among complications, second malignancies are the most serious. Second primary tumours are frequently found in patients with a history of head and neck malignancy. There is evidence of second neoplasms occurring among children previously treated for ALL with chemotherapy and radiation before the age of five years.[46,47] Early diagnosis of the second tumours is required, as delays can seriously affect therapy and survival.[48]

Conclusion

The overall dental management of children with malignancies aims at maintaining oral health. Thorough examination and dental treatment before cancer therapy initiation is ideal. Preventive and palliative care is often required to cope with the short-term side effects. Following therapy, close follow-up to moniter growth and development is important. As therapies improve, the team involved in the management of malignancies in children will need to expand to provide the patients not only with increased life expectancy, but also with improved quality of life.

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Mechanical properties of hypoplastic/ hypomineralised first permanent molar teeth

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ABSTRACT

Enamel is ectodermally derived and is produced by ameloblasts which differentiate from inner enamel epithelial cells of the dental organ. Any local, systemic or genetic factor that disrupts the very sensitive ameloblasts at any stage can cause enamel defects.

One of the most commonly [affected] tooth is the first permanent molar. Restoration of affected first permanent molar teeth is difficult and frequently these teeth require extraction. The aim of this study was to determine the mechanical properties of affected and unaffected enamel in first permanent molars affected with enamel hypoplasia and examine these teeth affected with a scanning electron microscope (SEM). Eight first permanent molar teeth affected with enamel defects (enamel hypoplasia) were embedded, sectioned and finely polished. Using the Ultra-Micro-Indentation System (UMIS) an array of indentations was conducted every 100 µm from the unaffected cervical enamel into the hypoplastic region cuspally. The hardness and modulus of elasticity of the enamel dramatically decreases significantly in the hypoplastic region to have mechanical properties similar to unaffected dentine. The SEM investigation shows that the enamel in the affected region is significantly more porous and is more disorganised than the unaffected enamel. This study suggests that the difficulty in restoring first permanent molars affected by enamel hypoplasia may be due to the varying mechanical properties within the enamel of an affected tooth.

Treatment of Patients with Cleidocranial Dysplasia

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ABSTRACT

Cleidocranial dysplasia (CCD) is an autosomal dominant osteochondrodysplasia with significant and characteristic dental abnormalities. The disease gene was mapped to chromosome 6p21 and mutations were found in *CBFA1*, a transcription factor that activates osteoblast differentiation. The most striking oral abnormalities are the delayed eruption of the primary dentition, delayed eruption of the permanent incisors and first molars and failure of the primary cuspids and molars to resorb and their permanent successors to erupt. This presentation summarises the current understanding of the phenotype of CCD. Management strategies for children and adolescents with CCD will also be discussed.

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The dental implications and management of children with bleeding disorders

Winning 2003 Undergraduate Essay
Margaret Shim, University of Western Australia

The dental management of children with bleeding disorders presents a unique challenge to the dental practitioner. Treatment of patients with bleeding disorders can be accomplished if the clinician has an understanding of the normal haemostatic mechanisms as well as of the common abnormalities that can affect the coagulation system. Appropriate preoperative planning and evaluation in collaboration with the patient's physician and/or haematologist will ensure that quality comprehensive dental care can be provided with minimal risks of complications occurring. An aggressive prevention-oriented program should be implemented at any early age to reduce the need for invasive dental treatment. Should dental intervention be required, emphasis should be placed on providing appropriate replacement therapy before the dental procedure, selection of conservative treatment approaches and use of local haemostatic measures to facilitate haemostasis.

The dental practitioner may also play a role in initial identification of child with an undiagnosed bleeding disorder when presented with uncontrolled prolonged bleeding episodes subsequent to a traumatic injury. Knowing when to refer the child to a medical specialist for investigative tests is important to ensure proper diagnosis.

The oral tissues present several physiologic concerns that increase the risk of postoperative bleeding in patients who have a coagulation defect. First, blood flow to the oral tissues is greater than the skin[1] and second; the oral mucosa exhibits significant fibrinolytic activity[2]. The clinical manifestations of disorders of haemostasis vary depending on the phase affected. Defects in primary haemostasis generally result in bleeding from mucosal surfaces, with the presentation of petechiae and purpura[3]. These disorders include von Willebrand's disease as well as defects in platelet function (see table 1). In contrast, defects in secondary haemostasis such as haemophilia (see table 2), results in bleeding that tends to be more deep-seated in muscles and joints[3]. Haemophilia, the most common of the inherited bleeding disorder is characterised by prolonged coagulation times due to deficiency of particular factors in the coagulation cascade[4]. In both disorders, uncontrolled prolonged oral bleeding can occur from innocuous insults such as a tongue laceration or cheek biting.

Characteristically, children with a decrease in number of platelets or platelet function will bleed immediately after trauma or surgery. Haemophiliac bleeds are delayed 12 to 24 hours as primary haemostasis is not impaired and local pressure has little effect[3]. These children may also present with spontaneous gingival bleeding and prolonged episodes of bleeding after minor trauma or tooth brushing.

Preoperative evaluation

Evaluation of children with suspected bleeding disorders require a complete medical history as well as laboratory tests. Before dental procedures are initiated, the parents should be asked specifically about previous episodes of haemorrhage that may have occurred after traumatic injuries, dental procedures and surgical therapy, as well as episodes of epistaxis[5]. In addition, the clinician should obtain information about the patient's family history and use of medications. Often the first indication of an undiagnosed coagulation defect in a child is uncontrollable, prolonged episodes of bleeding after a traumatic injury. In such cases, complete medical history, laboratory-testing requests in consultation with the patient's physician are necessary to arrive at a diagnosis.

Platelet disorders

Thrombocytopenia	Decreased platelet count	Idiopathic thrombocytopenic purpura Marrow suppression by drugs or haematological diseases such as aplastic anaemia Chemotherapy and radiotherapy
Thrombocytosis	Increased number of platelets ($>500x10^9$ per L).	Abnormal platelet function May present with myeloproliferative disorders
Platelet-function disorders	Decrease in platelet aggregation	Congenital or acquired Use of NSAIDs Metabolic diseases, such as Gaucher's disease
Connective-tissue diseases	Vascular abnormalities and capillary fragility (bruising and purpura)	Scurvy (vitamin-C deficiency) Ehlers-Danlos syndrome
Von Willebrand's disease	Decrease in platelet function	Disorder of coagulation

Table 1: Adapted from Cameron & Widmer. Medically compromised children. Chapter 8. In: A handbook of pediatric dentistry, 2nd ed. New York: Mosby 2003.

Coagulation disorders

	Aetiology & transmission	Laboratory findings	Clinical findings	Treatment
Factor VIII deficiency (Classic haemophilia, haemophilia A)	X-linked recessive inheritance, defect in antihemophilic factor (factor VIII)	Normal bleeding time Normal prothrombin time Normal thrombin time Prolonged partial thromboplastin time Normal platelet count Factor VIII assay 0% to 40%	Only males affected (clinical findings vary with severity of disease) Severe <1% factor VIII → spontaneous bleeding into joints and muscles, including intracerebral haemorrahage Moderate 2-5% factor VIII Less severe bleeding, usually follows minor trauma Mild 5-25% factor VIII, may not manifest until middle or old age after significant trauma or surgery	Replacement with plasma product containing Factor VIIIc Antifibrinolytic (EACA) Local and topical measures 15% pt from antibodies (inhibitors) to factor VIII, complicating management
Factor IX deficiency (Christmas disease, haemophilia B)	X-linked recessive transmission Deficiency in factor IX Defect in molecule identified in some cases	Same as above Factor IX assay 0% to 40% Elevated APTT	Same as above	Prothrombinex or fresh frozen plasma for mild cases.
Factor XI deficiency (haemophilia C)	Autosomal recessive inheritance	Normal bleeding time Normal platelet function tests Normal prothrombin time Prolonged partial thromboplastin time Abnormal thromboplastin generation test in severe cases Factor IX assay 0% to 40%	Prolonged bleeding after surgery or trauma Male and females affected	Replacement with fresh plasma or plasmapheresis Local and topical measures
Von Willebrand disease	Autosomal dominant inheritance Abnormality of von Willebrand factor (VWF)	Prolonged bleeding time Decreased platelet adhesiveness Decreased platelet aggregation with ristocetin Possible prolonged partial thromboplastin time if Factor VIII less than 30%	Hemorrhages from mucous membrane Easy bruising Menorrhagia Becomes milder with age Males and females affected	Type I treated with DDAVP Types II and III require cryoprecipitate.
Others disorders of coagulation Vitamin-K deficiency Liver disease Disseminated intravascular coagulation – overwhelming infections.				

Table 2: Modified from Stewart et al. Pediatric Dentistry: Scientific foundations and Clinical practice. St. Louis: Mosby, 1982.

Preventive Phase

The hallmark of dental management of children with bleeding disorders is prevention. An individualised preventive program incorporating proper toothbrushing and flossing techniques, appropriate topical fluoride exposure and adequate systemic fluoride as well as proper diet and frequent clinical examinations will reduce the need for invasive dental procedures[6,7,8]. Rubber cup prophylaxis and supragingival scaling can be safely performed without prior replacement therapy even in severe haemophiliacs as long as care is taken to minimise trauma to soft tissues[8.] In situations of minor haemorrhaging, local measures including direct pressure with moistened gauze squares can be used[8]. Topical application of thrombin has been advocated for minor haemorrhaging that persists for several minutes[9]. Topical thrombin acts by directly converting the fibrinogen in the blood to fibrin[5].

If subgingival calculus is evident in conjunction with soft hemorrhagic tissues, replacement therapy before scaling should be considered[10]. If such a situation arises, the scaling and polishing should be performed in one visit to minimise the need for multiple infusions[11]. According to some authors[8,12], patients who require deep scaling should be initially scaled supragingivally. The tissues should be allowed to heal for 7 to 14 days, during which time gingival shrinking occurs, as oedema and hyperaemia diminish. Subsequent treatments to remove calculus and irritants will therefore incur less risk of bleeding from the tissue[8]. Other authors have suggested quadrant therapy in mild or moderate haemophiliac patients to circumvent the need for factor coverage and permit the use of local measures such as pressure or topical agents[13]. Alternatively, ultrasonic instruments have been advocated for professional oral hygiene in order to minimise trauma and accidental tissue laceration[14]. It is imperative that patients requiring periodontal therapy are placed on a maintenance schedule to ensure optimal periodontal health.

Both factor replacement and antifibrinolytic therapy is indicated prior to frenectomy procedures or other periodontal surgery[8]. If large amounts of bleeding are anticipated, these procedures should be done in a hospital environment, subsequent to consulting with the attending physician or haematologist to determine the factor correction required and the need for postoperative management. Electrocautery is generally not recommended for a haemophiliac patient due to the possibility of spontaneous haemorrhaging several days after the procedure when clot lysis normally occurs[8].

Routine dental care

In reference to routine dental care, most children with bleeding disorders can be treated on an outpatient basis depending on the complexity of the treatment, the behaviour management of the child and whether all prerequisite preparation in regards to their medical condition is achieved. Treatment is preceded by taking a thorough medical history, which includes past episodes of bleeding, and the severity and location of those episodes. Information regarding the invasiveness of the dental procedure and the degree of bleeding anticipated, as well as the time frame necessary in oral wound healing should be provided to the haematologist to determine the need for factor replacement therapy. Appointments should be arranged so that maximum treatment per visit can be completed to keep the number of factor infusions to a minimum. When feasible, appointments should coincide with the patient's scheduled time for factor replacement[10].

Pain control

Due to the risk of haematoma formation, intramuscular injection of hypnotic, tranquilising or analgesic agents is contraindicated. However, sedation or nitrous oxide-oxygen inhalation may be considered where significant patient apprehension is evident. Acetaminophen compounds (Tylenol®, Tempra®) are appropriate for management of acute pain of moderate intensity[7,8]. Analgesics containing aspirin or anti-inflammatory agents (eg ibuprofen) are contraindicated as they alter platelet function[10]. For severe pain, narcotic analgesics are recommended and are not contraindicated in the haemophiliac patient[8].

Local anaesthesia

Maxillary infiltration anaesthesia can generally be administered without pre-treatment with antifibrinolytics and/or factor replacement. However, factor replacement of 40% activity levels is recommended if the infiltration injection is into a highly vascular area or loose connective tissue[15]. Periodontal ligament injections may be administered without prior factor replacement. The solution is placed under moderate pressure along the four axial surfaces of the tooth by placement of the needle into the gingival sulcus and periodontal ligament space[16]. However, some authors argue that the anaesthesia produced by intraligamentous injections is not profound enough or of long enough duration for restorative procedures[17].

Caution is mandatory when considering block anaesthesia due to the potential for complications to arise. The highly vascularised loose connective tissues at the sites of inferior alveolar nerve injection and the posterior superior alveolar nerve injection pose the threat of forming a deep-seated hematoma which has the potential of obstructing the airway and creating a life-threatening bleeding episode[8,18]. Block anaesthesia requires preoperative factor correction of 40% to prevent hematoma formation[8,10]. Slow atraumatic injections with proper bevel orientation and frequent aspiration are critical in minimising postoperative problems. In the event of a bloody aspirate, further factor replacement will be required and the attending physician should be notified immediately following the operative procedure. Subsequent to administration of local anaesthesia, all patients with bleeding disorders should be observed for the development of a hematoma and immediately referred for treatment in the event of hematoma formation[5].

Antifibrinolytics

Antifibrinolytics such as epsilon amino caproic acid (Amicar®) or tranexamic acid (Cyclokapron®) may be used as specific adjunctive therapies to help control oral bleeding during dental treatment of patients with bleeding disorders[8]. Epsilon amino caproic acid (EACA) retards early clot dissolution by acting against plasminogen activators in subendothelial areas of the oral

mucosa[18]. Antifibrinolytics may also be administered alone without factor replacement if minimal bleeding following certain dental procedures is anticipated.

In children, EACA is administered immediately prior to dental treatment in an initial loading dose of 200 mg/kg by mouth[8]. Subsequently, 100 mg/kg of EACA is administered orally every six hours for five to seven days. The advantage of EACA for children is that it is available in both tablet and liquid form. The dosage for tranexamic acid is 25 mg/kg given immediately prior to dental treatment[8]. The same dosage is continued every eight hours for five to seven days postoperatively. Tranexamic acid is available in tablet form. The advantage of tranexamic is a smaller dosage and longer intervals between doses[8]. The use of these agents decreases the number of factor infusions necessary[19]. According to some authors, minimising the use of factor replacement therapy is advocated whenever possible as it decreases the potential for contracting hepatitis B, hepatitis non-A/non-B, delta hepatitis, or Acquired Immunodeficiency Syndrome (AIDS)[6,19].

Common side effects associated with the use of antifibrinolytics include headache, nausea and xerostomia. According to McKown and Shapiro[8], these side effects are usually tolerable and unless severe, do not require discontinuation of the medication. Antifibrinolytics should not be used when renal or urinary tract bleeding is present or when there is any evidence of disseminated intravascular coagulation, as they may lead to thrombosis[8].

Restorative procedures

Most restorative procedures on deciduous teeth can be successfully completed without factor replacement using periodontal ligament injections or local infiltration[8]. Small lesions may be restored using nitrous oxide oxygen inhalation analgesia alone. The use of acetaminophen with codeine may also help to decrease discomfort in the child[8].

Certain precautions taken during restorative procedures will greatly reduce the hazard of excessive bleeding[6,7]. Rubber dam isolation not only guards against accidental aspiration of foreign objects but also minimises the potential of lacerating the gingiva, tongue, buccal mucosa and lips. These soft tissues are highly vascular and their accidental laceration could present a difficult and dangerous management problem. Thin rubber dam is preferred because there is less tendency to torque the rubber dam retainer and abrading the gingival tissues[20]. Retainers should be carefully placed so that is does not traumatise the gingival tissues. Retainers with subgingival extensions such as the 8A or 14A should also be avoided. Interdental wedges and matrices can be used conventionally to retract papillae from the operative site. Judicious use of high-speed suction and saliva ejection is necessary so that sublingual hematomas are not created. Care must also be used in placement of intraoral radiographic films, particularly in highly vascular sublingual tissues. Careful placement of retraction cord before preparation of a subgingival crown (eg SSC) will minimise trauma to gingival tissues. Periphery wax used on the impression tray aids in preventing possible intraoral laceration during tray placement. The use of pit and fissure sealants is advocated as a non-invasive preventive procedure to decrease the need for extensive restorative dentistry[10].

Pulpal therapy

Pulpal pathosis can be treated routinely in children with bleeding disorders. Rather than extract a carious tooth, pulpotomy or pulpectomy procedures are preferred to prevent complications occurring. Most vital pulpotomy and pulpectomy procedures can be successfully completed using local infiltration anaesthesia[8] and/or local haemostasis with pressure or chemical agents[18]. Nitrous oxide-oxygen inhalation analgesia may also help to alleviate any discomfort[8]. If the nerve of a vital tooth is exposed, an intrapulpal injection may be safely used to control pain. Haemorrhaging from the pulp chamber can be readily controlled with pressure from cotton pellets[8]. If pulp tissue is necrotic, local anaesthetic is usually unnecessary. When endodontic therapy is performed, careful instrumentation and filling will prevent overextension beyond the apex of the root of the tooth and resulting in periapical bleeding[18].

Oral surgery management

Oral surgery can be performed on an outpatient basis provided that a facility is available for the patient to receive infusions if home infusion is not feasible and a coagulation laboratory that can perform the necessary laboratory evaluations is accessible. However, in young children, general anaesthesia in a hospital setting may be necessary due to the complexity of treatment and/or behavioural management problems. The main concern with general anaesthesia is that any tracheal intubation or laryngeal mask may induce laryngeal hematoma and nerve trunk infiltration could produce lateropharyngeal hematoma[21]. These forms of hematoma are very difficult to control and could lead to upper airway obstruction.

Prior to the surgical procedure, the dentist should inform the haematologist of the degree of surgical trauma anticipated and the duration of the healing process so that the haematologist can then determine the appropriate amount and duration of factor concentrate replacement required for surgery and postoperative management. For simple extractions of erupted permanent teeth and multirooted teeth, a 40 per cent factor correction is administered intravenously on the day of surgery to raise the level of the deficient factor[8]. Due to the exposure to factor concentrate, all extractions should be completed in one appointment. Antifibrinolytic therapy should be started immediately prior to the procedure and continued for seven to ten days postoperatively.

Absorbable haemostatic agents that have been advocated to aid in local haemostasis after extractions include Gelfoam, oxidised cellulose (Oxycel®), Surgicel® and Avitene®. Gelfoam is a water-insoluble gelatin sponge that acts by disrupting the platelets and establishes a framework with fibrin to create a clot[5]. The oxidised cellulose preparation, Oxycel® and the oxidised regenerated cellulose preparation, Surgicel®, have an affinity for haemoglobin that leads to an artificial clot[5]. Avitene®, a microfibrillar collagen haemostat, is useful in the oral environment as it can be rapidly moulded to the required size and shape or spread over a bleeding surface as it

adheres to moist surfaces[5]. It appears to provide a fibrillar mesh in which platelets become physically entrapped, aiding in aggregation and chemical reactions necessary for hemostasis[5]. The sockets should be packed with Gelfoam, followed by sprinkling of microfibillar collagen or topical thrombin[8]. Direct pressure with gauze should then be applied to the area. Stomahesive may be placed over the wound for further protection from the oral environment. Sutures may increase bleeding and therefore are not recommended unless they are needed to close a mucoperiosteal flap[4, 22], or will markedly enhance healing[8]. Resorbable sutures are recommended when suturing is necessary[8].

Specific postoperative instructions should be given to the child and parent. Postoperative diet management is directed toward preventing clot injury. The child should be placed on a clear liquid diet for the first 72 hours. For the next week, a soft, pureed diet is recommended. During this time, the patient should avoid using straws or metal utensils, pacifiers or bottles, to prevent dislodgement of clots formed. After ten days, the patient may begin to consume a more normal diet.

For surgical extractions of impacted, partially erupted or unerupted teeth, a higher factor activity level preoperatively and infusions postoperatively may be necessary[8]. This is due to the greater degree of surgical trauma and longer healing period anticipated. Antifibrinolytic therapy should also be started immediately prior to the procedure and continued for seven to ten days. For simple extractions of single rooted primary teeth (ie incisors and canines) one must evaluate the amount of root development present to determine whether factor replacement therapy is necessary prior to extractions. If there is complete root development, factor replacement therapy may be necessary. If there is only partial root formation, antifibrinolytic therapy along with local hemostatic agents and pressure may be all that is required.

Surgical complications

The patient should be monitored closely for any bleeding episodes postoperatively and when it occurs, it should be treated aggressively and without delay. Bleeding tends to occur three to four days postoperatively when the clot begins to break down. Both systemic and local treatment must be used for control of haemorrhage. Additional replacement with factor concentrate and/or EACA is used to control any recurrent bleeding. Following adequate replacement with factor concentrate, the clot should be removed and the area cleansed to help isolate the source of bleeding[8]. The socket should then be repacked.

Exfoliation of primary teeth

The normal exfoliation of primary teeth usually does not require factor replacement and bleeding that occurs can usually be controlled with direct finger and gauze pressure maintained for several minutes[8]. The direct topical application of hemostatic agents such as thrombin or Avitene® may also help with local hemostasis. If there is continuous slow bleeding, antifibrinolytic therapy may be started.

Orthodontic treatment

Orthodontic treatment of children suffering from bleeding disorders does not present a significant problem. Early recognition of an orthodontic problem is important as early intervention can eliminate the need for complex treatment later on. Both interceptive and full-banded orthodontics may be performed.

Professional care in appliance design and execution will prevent incidental gingival or mucosal trauma. Protruding sharp edges and wires should be eliminated and bands should be carefully adapted into place to prevent lacerating the oral mucosa. Bleeding caused by an accidental scratch or minor laceration of the gingiva usually responds to applied pressure for five minutes[9].

Serial extractions will require interdisciplinary management with consultations between the orthodontist, surgeon, and haematologist. Oral hygiene compliance during orthodontic treatment is critical to prevent inflamed, oedematous and hemorrhagic gingival tissues.

Dental emergencies

Oral trauma is a common occurrence during childhood. Management of bleeding injuries, including hematomas, in the mouth of the haemophiliac patient may require a combination of factor concentrate replacement therapy, antifibrinolytic therapy and treatment with local hemostatic agents.

Conclusion

Management of children with bleeding disorders requires an interdisciplinary approach whereby collaboration with the patient's physician and/or haematologist will ensure that dental procedures can be performed with minimal risk to the physical health of the patient. Emphasis should be placed on early preventive intervention, preoperative evaluation and postoperative management, selection of conservative treatment approaches, meticulous surgical techniques and the use of local haemostatic measures to aid haemostasis.

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Association between Previous Illness, Medication Use and Early Childhood Caries

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ABSTRACT

Objectives: To investigate the association between previous illness (>1month), medication use (>1month), parental request for sugar-free medication and early childhood caries (ECC) experience within a regional preschool child population.

Methods: A cross sectional sample of 2,515 children aged 4-5 years were examined in a community setting and ECC experience was recorded by percentage of children with dmft≥1, anterior (anterior dmfs≥1) or posterior (anterior dmfs=0) caries pattern and non-severe (dmft<6) or severe (dmft≥6) caries form. A self-administered questionnaire obtained information regarding previous illness and medication use from a parent or guardian. Caries prevalence, pattern and severity in children with previous illness (439), medication intake (459) and request for sugar free medication (134) were compared with the remainder of the sample. Medication intake was further subdivided into bronchodilator, antibiotic, anticonvulsant, antidepressant and reflux groups. The data were analysed using Chi-square and analysis of variance procedures at $p < 0.05$ level of significance.

Results: Previous illness and medication intake were significantly associated with caries severity ($p = 0.003$, $p = 0.03$ respectively). Parental request for sugar-free medication was not associated with ECC experience at 4-5 years. Previous bronchodilator use was significantly associated with anterior caries pattern ($p = 0.009$) and caries severity ($p = 0.005$).

Conclusions: Previous illness and medication use are significant risk factors for the development of ECC in a preschool child population. In particular, severity and pattern of ECC was significantly influenced by a previous respiratory illness and the use of a bronchodilator within the group of medically compromised children.

Langerhans' Cell Histiocytosis: A case report and literature review

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ABSTRACT

Langerhans cell histiocytosis refers to a spectrum of rare proliferative disorders of Langerhans' cells. It encompasses three disease patterns: Eosinophilic granuloma, Hand-Schüller-Christian disease and Letterer-Siwe disease. The aetiology is unknown and affects mainly children and young adults. Langerhans' cell histiocytosis has a marked variability in clinical presentation. The granulomatous infiltrations commonly involve bone, skin, lung, liver and pituitary tissue. Oral lesions have been reported in up to 77% of the affected population. These include ulceration of the oral mucosa, bone resorption and 'floating' teeth. The following is a case report of a young girl with the above condition.

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ANZSPD – Branch News 2004

New South Wales

Now that the year has started, the NSW committee is gearing up for another busy year. All members in NSW are looking forward to the Biennial meeting in Melbourne in March. The programme looks fantastic although most members are anticipating that the social events will again be the highlight. As the ANZSPD biennial meeting is early in the year, there are three meetings planned for the year where we hope to have both local and interstate speakers backed up by the second year post graduates at the University of Sydney, Drs Fiona Bell and John Camacheo.

Organisation is well underway for the IAPD meeting in 2005 with the planning of the scientific and social programmes well underway. Members should be receiving information about this over the coming year.

Happy 2004 from all NSW ANZSPD Branch Members.

Erin Mahoney

Queensland

The Branch has had another active year with regular meetings and lectures. At the March meeting Dr Jim McGill, metabolic physician, spoke on "Modes of Inheritance and some of the more common types of hereditary disorders with dental manifestations". In May, Dr Chris Ho, orthodontist, discussed "Orthodontic teaching and problem solving in general practice".

Our annual study weekend was held in August at the Grand Pacific Resort in Caloundra. The theme of the meeting was Special Care in Dentistry. Our keynote speaker was Dr Chris Olsen, Federal President of the Society. There were a variety of other presentations from a number of guest speakers as well. Attendees were given a first class level of professional information on treating children with special needs including management techniques and implications for Paediatric Dentists.

The President's At Home was held at Tattersall's Club in November. Dr Peter Farrington spoke about his recollections during 20 years as a dentist in Thailand. Members and their partners thoroughly enjoyed the evening. The annual general meeting will be scheduled for February 2004.

The support of our sponsors, Oral B / Gillette and 3M ESPE, is as always very much appreciated. The valuable assistance of Dr Matthew Fracaro, Secretary/Treasurer, and Dr Kerrod Hallett, Federal Councillor, as well as the unending support of the membership throughout the year are also acknowledged.

Here's looking forward to another rewarding year in 2004.

Laurie Bourke

South Australia

This year we had four branch meetings that covered a wide variety of subjects.

At the AGM Dr John Kibble spoke of his experiences in East Timor. John was a part of a group that visited East Timor to assess the Dental Nurse Training Programme.

Post independent East Timor is certainly very grim, with poor communication, lack of power and proper sanitation. Dentist and dental nurse numbers are very low compared to the population they must service. Dentists undergo a six year training programme and learn how to do extractions and restore teeth. However, their main role is administrative. The Dental Nurses, on the other hand, do a three year training programme and are taught basic dental care and extractions (mainly). There are big shortages in relation to dental equipment and materials. Treatment of any type is hampered by these shortages as well as continuing problems with lack of a constant power supply, water etc. In fact all the basic necessities that we take for granted. John also discussed briefly the epidemiological studies done by Professor John Spencer.

This year the programme also included a visit from the current Federal President, Dr Chris Olsen. Chris spoke to us about Dental Trauma, which is always of interest and also discussed current Federal matters with members at the meeting.

The August meeting looked at the role of GIC's in Paediatric Dentistry. Ms Trish Hogan from GC gave an excellent presentation on the use of GIC's and also discussed Tooth Mousse. This was followed by a "tasting". Everyone agreed the flavours were most agreeable and would be acceptable to children who don't usually like strong flavours. At this meeting Dr Margaret Evans also spoke about her experiences working as a volunteer in Guatemala. Margaret has previously visited Vietnam and India as a volunteer with Rotary. Although a little bit better off than those in East Timor the delivery of dental care was still very basic, despite the fact that this programme has been ongoing for many years.

We ended the year with a meeting in late November that doubled as a Christmas celebration. Drs. Sam Gue and Scott Smith gave excellent case presentations and all the members enjoyed this plus a very pleasant Indian Banquet and some Christmas Cheer.

In all 2003 was a successful year for the SA Branch and we are all looking forward to 2004, with quite a few members making the trip to Melbourne in March 2004.

Sue Springbett

Western Ausralia

The final meeting for 2003 was held at A.D.A. House in West Perth. It was in the form of a half day course and featured Dr Sally Hibbert as the main speaker. Sally had flown over to Perth to see her beloved English Rugby Union team do battle with the South African team, and so it was a golden opportunity for her to deliver an update on the subject which was covered by David Manton's literature critique in the previous edition of Synopses.

The topic, "Why Save Primary Teeth" was both eye-opening and thought provoking. It is quite obvious, in this litigious age, that such an approach cannot really be countenanced, but at

the same time, it did draw to our attention the shortcomings of certain treatment options and how these can be dictated by prevailing publicly funded health systems. The support speaker was Dr Peter Dillon, a Perth orthodontist, who presented an easily followed talk on Space Maintenance. The Annual General Meeting of the Branch was due to follow this meeting, but had to be postponed until Branch President, Tim Johnston, returned from the IAPD meeting in New Orleans.

At the reconvened Annual General Meeting, Tim Johnston was re-elected as President, and your correspondent, Alistair Devlin re-elected as Secretary – Treasurer. John Winters remains as Federal Councillor. Peter Dillon, Kate Dyson, Jeremy Foster, Mark Foster, Theo Gotjamanos, Peter Gregory, John Hands and Peter Readman were elected as Committee members.

The first meeting for 2004 will be on 5th March when IAPD Secretary-General, Professor Gerry Wright and his wife, Nancy will be the guests of the Branch at a dinner meeting to which members and their partners will be invited. Gerry and Nancy will be making a joint after dinner presentation.

Professor Wright has been awarded the 2004 A.J. Herman Fellowship by the University of Western Australia, and will be spending a fortnight in Western Australia. During that time, he will deliver a number of lectures to undergraduates, he will participate in clinics at the Oral Health Centre of WA and at the Princess Margaret Hospital for Children, and will deliver the eponymous A.J. Herman Lecture to the wider University community.

Alistair Devlin

New Zealand

We were all very pleased with the attendances and contents of Dr Seow's lectures – we had much positive feedback and as a result we are grateful to all responsible for her tour. The last quarter has seen the branch in holiday mode.

Our Labour Government is now in its second term of power. It has adopted a consultative approach to the development of health care policy. The ANZSPD (NZ Branch) has been involved in three important consultations. Firstly, the executive made a submission on the NZ Accident Compensation Commission (ACC) Review with respect to dental care for children who have suffered orofacial trauma.

Secondly, we have had representation on a government committee charged with developing standards of care for children receiving hospital care. It is envisaged that once this document has been completed standards may be expanded to include other types of health care for children. The draft document recommends that children should be examined and cared for by professionals with recognised qualifications in paediatrics. And that the parent-child relationship is central to the care of the child and as such parents need to be accommodated to ensure that this can be achieved. Dr Dorothy Boyd is representing the ANZSPD (NZ Branch) and the Society of School and Community Dental Services on this committee. I filled in for one session, and it was a great opportunity to be amongst so many people interested in delivering high quality health care to children, and to realise that we are all often experiencing similar problems across varied disciplines.

Thirdly Dr Joanna Pedlow is representing the ANZSPD (NZ Branch) on a government committee which is developing standards for Daycare Surgery for children.

On a more personal note Dr Katie Ayers has shifted with her family from Dunedin to Hamilton. We hope that other branch members have enjoyed a summer break with their families.

MaryAnne Costelloe
Stratford,Taranaki

ANZSPD Federal Secretary-Manager's Report

1. As readers will see elsewhere in this edition, the ANZSPD Post-graduate and Under-graduate Essay Competitions for 2003 have been judged. The winners were Yaso Ramadas from Melbourne in the Post-graduate Competition and Margaret Shim from Perth in the Under-graduate Competition. Once again, the judges have commented on the uniformly high standard of the entries in both competitions.

2. The Federal Council of ANZSPD will be meeting on Wednesday, 17 March 2004 in Melbourne, just before the Federal Convention gets underway. Provincial branches have been asked to submit items for the agenda – this is in addition to the usual items of Presidential, financial and Synopses reports, and of course, the all important report on progress of the Sydney 2005 IAPD Congress from Congress Chairman, Richard Widmer. Another important item will be the election of office bearers by the Councillors. The new Executive will be installed at the General Meeting of the Society, which will be held at 4.00 pm on Saturday, 20 March 2004.

3. Anybody who has had anything to do with the organisation of the Sydney 2005 IAPD Congress will know the enormous job it is and how the maximum time is required to get everything arranged. IAPD, at their meeting in New Orleans in October 2003, was supposed to appoint a nation to hold the 22nd Congress in 2009. The IAPD Council considered no bid was satisfactory and has now called for new bids. The final decision won't be able to be made until the Sydney meeting in 2005. Four years to prepare will just have to be enough for the successful bidder.

4. Members who have taken advantage of the chance to take up individual IAPD membership at the same time as their ANZSPD membership probably will have received a notice from the IAPD Secretariat. This notice will offer renewal of their IAPD membership. It is entirely up to the individual to renew the IAPD membership as offered or do it with their ANZSPD membership as before.

Alistair Devlin

Management of natal and neonatal teeth
Two Case Reports

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MDSc candidate in Paediatric Dentistry, School of Dental Science, University of Melbourne, and Honorary Dental Officer, Royal Children's Hospital of Melbourne, Australia.

Abstract

Two cases are presented of natal and neonatal teeth preventing adequate feeding in neonates. In the first case, a natal tooth prevented establishment of adequate suckling in a two day old infant; while in the second case, two neonatal teeth, which erupted at six days, had increasingly disrupted bottle feeding in an infant who was five weeks old at presentation. Active early management of natal or neonatal teeth is required only if they prevent satisfactory suckling, cause traumatic ulceration to adjacent soft tissues, or if marked mobility presents a theoretical risk of inhalation. Consideration is given to alternative management strategies and associated pitfalls. In these two cases, extraction quickly allowed establishment of breast feeding, and resumption of bottle feeding respectively, which were the chief concerns at presentation.

Introduction

Natal teeth may be defined as teeth which are present in the oral cavity at birth, whereas neonatal teeth may be defined as teeth erupting during the neonatal period from birth to 30 days.[1]

Formerly, natal and neonatal teeth have been described as congenital teeth, foetal teeth or dentitio praecox.[1-3] None of these terms describe the aetiology, morphology or location of such teeth. In a review of literature, Zhu and King noted a widely varying prevalence, from 1 in 11 to 1 in 30,000 live births, with the majority of studies reporting between 1 in 700 to 1 in 6,000.[3]

Most natal and neonatal teeth occur in the anterior region of the mandible, either singly or in pairs.[3] Most natal and neonatal teeth are early erupting members of the normal primary dentition, although an incidence of supernumerary teeth varying from 1 to 10 per cent has been reported.[3,4] Natal and neonatal teeth may be attributed to a superficial positioning of the developing tooth germ which predisposes to early eruption.[3,4] A number of studies have suggested a familial trait for this anomaly.[3,4]

Reported problems from natal or neonatal teeth requiring dental intervention include difficulties in suckling, either by pain and/or sublingual Riga Fede's ulceration experienced by the infant, or discomfort to the nursing mother.[3-5] Perceived risk of inhalation of an excessively mobile tooth, abnormal morphology or supernumerary classification are also reasons cited for active management.[4,6]

Case report

Case 1

A two day old male Caucasian infant was referred by the attending pediatrician, who prior to contacting one of the authors (CBO), had made several unsuccessful attempts over the previous 24 hours to obtain the services of a paediatric dentist. The presenting complaint was of breast feeding problems due to pain resulting from nipple biting by the natal tooth. The baby was described by the paediatrician as "going through the right motions of suckling". There was no family history of natal or neonatal teeth.

The child was born in a major metropolitan medical teaching hospital having both midwifery and neonate intensive care facilities. Delivery was by Caesarian section, at 39 weeks from a prima gravid mother. The child, otherwise healthy, weighed 3.95 kg, and had an Apgar score at one minute and five minutes of eight and ten respectively. Vitamin K had been administered soon after birth, which is usual practice for new born infants in Australia as a preventive against vitamin K deficiency bleeding.[7] The infant was brought to the author's private practice located 800 metres from the hospital in a pram by a mid wife, and accompanied by a somewhat overwhelmed new father.

On examination, the infant was found to have a moderately mobile tooth resembling a primary mandibular central incisor just to the left of the lower midline (Fig.1 far right). The father was informed that the tooth was most likely a primary mandibular incisor tooth that had erupted prematurely, or less probably a supernumerary tooth. The father was informed also that loss of a mandibular primary incisor had not been shown to cause crowding problems, although drifting of adjacent teeth would probably occur. The father then agreed to removal of the natal tooth.

The natal tooth was extracted following administration 0.2ml of 2% Xylocaine® (Astra Pharmaceuticals Pty Ltd, North Ryde, NSW, Australia) with

1 in 80,000 adrenaline infiltration local anaesthesia, with the infant's head being supported in the pram. Haemostasis was quickly achieved. Several days later the infant's mother reported that breast-feeding had been established immediately and satisfactorily on the infant's return to her.

The extracted natal tooth had little root formation, which explained its mobility (Fig.2 right). The tooth was later submitted for biopsy (Fig.3 right). Microscopic examination showed a tooth crown with hypomineralised enamel matrix, and normally developing dentine and dental pulp.

Case 2

A healthy five week old male Caucasian was presented to the Royal Dental Hospital of Melbourne with a complaint of not feeding adequately due to two loose teeth in the anterior region of the mandible (Fig 4 below). The parents stated that the neonatal teeth had increasingly disrupted satisfactory bottle-feeding, following their eruption at six days. Bottle-feeding had been introduced due to an inadequate maternal milk supply. The infant otherwise had no health problems, and had received vitamin K prophylaxis at birth.[7] There was no family history of natal or neonatal teeth.

On examination, two extremely mobile teeth resembling primary mandibular incisors were present in the anterior region of the mandible. The parents were informed that these teeth were probably either early erupting primary mandibular central incisors, or less likely were supernumerary teeth, for which removal was indicated. Both parents then requested extraction.

The baby was positioned in the pram, and the head was stabilised while both teeth were extracted following administration of 0.2 ml of Xylocaine® with 1 in 80,000 adrenaline infiltration local anaesthesia. Haemostasis was quickly achieved. The extracted neonatal teeth had no root formation. Bottle-feeding was reestablished immediately and satisfactorily prior to the infant being dismissed.

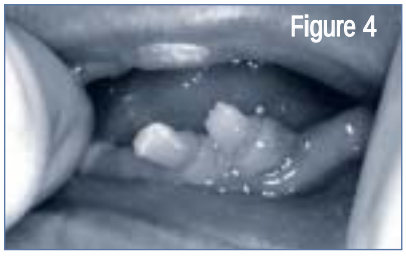

Figure 4. Case 2, neonatal teeth at presentation.

Discussion

Active management of natal teeth is necessary only if they are causing a clinical problem. Removal is not indicated merely because they are mobile, unless mobility is excessive. The risk of inhalation of loose natal or neonatal teeth seems imagined rather than real, as no case of inhalation of loose natal or neonatal teeth has ever been reported in the dental literature so far as the authors are aware. Natal and neonatal teeth often become firmer over time as the root develops.[4,6]

Active management usually involves extraction, or more rarely rendering the teeth smooth to oral tissues and nipple, either by the use of enamel-bonded composite resin, or by periodic application of wax like material over the teeth.[3-6,8,9] In Case 1, extraction of a natal tooth which was preventing breast feeding, quickly and expeditiously allowed establishment of satisfactory breast feeding.[9] In Case 2, the removal of two neonatal teeth immediately led to a resumption of satisfactory bottle-feeding.[9]

In Case 1, alternative management strategies may have been to commence bottle-feeding in lieu of breast feeding, or perhaps to attempt smoothing of the natal tooth. Smoothing of a natal tooth with Stomadhesive Wafers® (E.R. Squibb & Sons, Noble Park, Victoria, Australia), a wax like material, prior to each feeding, or smoothing of the tooth with an enamel bonded splint, thereby rendering the tooth less liable to cause trauma is theoretically possible.[5,8]

Bottle feeding in new-born infants has obvious drawbacks, and incurs loss of the many well known benefits of breast feeding.[10-12] In Case 1, the use of Stomadhesive Wafers® to cover the tooth prior to each feed would have been a tedious and imperfect procedure, needed for an undetermined period of time. Smoothing of the tooth with an enamel bonded composite domed splint would be fraught with the difficulty of placing this in a newborn child, and on tooth enamel of likely poor bondability. However the

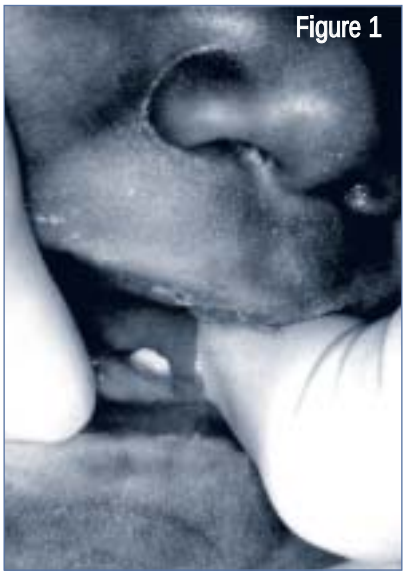

Figure 1. Case1, natal tooth at presentation.

Figure 2. Case 1, natal tooth after extraction.

Figure 3 Case 1, histology of decalcified natal tooth section.

technique of smoothing a tooth, whether by use of stomadhesive wafer or an enamel bonded composite domed splint has been shown to allow retention of teeth causing sublingual soft tissue Riga Fede's ulceration.[5,8] In Case 2, the extreme mobility of both neonatal teeth precluded any treatment option except extraction.[9]

Immediate risks of dental extraction in neonates include haemorrhage, or overdose of local anaesthetic if used injudiciously. Careful observation for any bleeding, and administration of vitamin K to newborn infants should reduce the risk of haemorrhage. Care must be taken to avoid use of excessive quantities of local anaesthetic. Using the limit of 6mg/kg of body weight of lignocaine with adrenaline vasoconstrictor would limit the quantity of lignocaine used to approximately 22mg, or one half of a 2.2ml carpule of Xylocaine.[13,14] In view of the immature liver metabolism of newborn infants, it would seem prudent to reduce this amount still further.[14,15]

In the long term, the loss of natal or neonatal teeth, if they are supernumerary teeth or belonging to a predeciduous dentition, will be of benefit to occlusal development. Removal of natal or neonatal teeth, which are early erupting members of the normal primary dentition, has not been shown to cause or aggravate later crowding problems in the mixed and permanent dentitions.[6,9,16] No unequivocal evidence has been published as to whether early loss of primary incisor teeth, either before or after eruption of the primary canine teeth, causes or aggravates crowding in the mixed or permanent dentitions. Published evidence of space loss occurring after premature loss of primary incisor teeth has been anecdotal and controversial, with no controlled studies having been reported, so far as the authors are aware.[6,9,16]

In both of the present cases, extraction of the natal tooth and neonatal teeth respectively, was the indicated treatment even though it was not known with certainty whether they were either prematurely erupting primary incisor teeth or supernumerary teeth.[17] Radiographs were neither indicated, nor taken, as any additional diagnostic information derived from them would not have changed the treatment plan.[17] Consideration of the "ALARA" (as low as reasonably achievable) principle of radiation hygiene, especially in such young patients, together with the clear cut need for extraction in these two cases, indicated that additional radiographic diagnosis would have been of no demonstrated net benefit to these infants.[17,18]

Extraction of natal or neonatal teeth must be performed carefully, ensuring that all odontogenic remnants are removed. Incomplete tooth removal has occasionally been reported, leading to continued growth of the dental papilla, and subsequently a second procedure to remove the remaining dental tissues.[19]

In Case 1, the difficulty of the attending paediatrician within a major teaching hospital to obtain easily assistance from dental colleagues is lamentable. Ideally, all medical teaching hospitals should have an attached dental clinic, or at the very least formalised access to dental advice and assistance. This report highlights that dental professionals need to develop closer working relationships with their medical colleagues.

Acknowledgement

In case 1, the role of Dr Mala Desai in initial liaison with the referring pediatrician is acknowledged.

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A clinical and molecular genetic study of oligodontia in three Australian families

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ABSTRACT

Background

The ectodermal dysplasias are a large and complex group of inherited diseases comprising over 170 different pathological clinical conditions. These conditions all share in common anomalies of the hair, teeth, nails and sweat glands and many are also associated with anomalies in other organs and systems.

These ectodermally derived anomalies are extremely variable in their presentation and clinical overlap occurs among the majority of ectodermal dysplasias. To date, few causative genes have been identified for these diseases to permit a more definitive diagnosis. From a dental perspective, the main clinical feature of oligodontia is not diagnostic in itself of ectodermal dysplasia as it may also manifest in other conditions or present as an isolated clinical trait.

Purpose of the study

1. To better differentiate the clinical features that are consistently associated with milder forms of ectodermal dysplasia from those associated with oligodontia as an isolated trait
2. To confirm these clinical findings by conducting genetic linkage analysis on members of several families who display dental characteristics associated with either oligodontia or milder forms of ectodermal dysplasia

Materials and Methods

The patients selected from this study had been previously seen through one of the three Departments of Paediatric Dentistry in Sydney, Australia and had been given a provisional diagnosis of ectodermal dysplasia or oligodontia by either a consultant paediatric dentist or clinical geneticist. In total, 26 patients from three families took part in the study which involved a genetic consultation, physical examination of the skin and epidermal appendages as well as an oral examination, salivary function test and hair and blood sample collection. DNA was then extracted from the blood samples in preparation for analysis of genetic linkage to the most likely candidate gene based on the clinical findings and the available genetic literature.

Preliminary Results

Linkage analysis was conducted on one large pedigree that displayed classic features of a mild form of ectodermal dysplasia. Linkage was not established with the candidate gene and a genome-wide scan is currently underway to determine whether an as yet previously unassociated gene shows linkage to the disease. Two other families with clinical features of oligodontia as an isolated trait have been characterised.

Colgate® Corner

by Dr Jackie Robinson
Colgate Professional Relations Manager

By the time this edition of Synopses reaches you, the 14th Biennial ANZSPD Conference will be a memory. Colgate was very pleased to again act as the Principal Sponsor for the conference and we congratulate the organising committee on the success of the conference. As always the conference was a wonderful blend of science and camaraderie.

For those members of ANZSPD who attended the conference, you would have seen and heard all the good news Colgate has about new toothbrushes for children – a strong compliment to Colgate's range of pastes for children. There are the new 3D Looney Tunes brushes – both manual and battery powered. And the exciting new Lego and Bionicles brushes.

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  The official language will be English. During the symposia, simultaneous translation to Spanish will be provided.

  www.eapd.gr for registration, abstract submission, and other information.

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